the aims stated is desirable and I sincerely hope it gets large support.—I am, etc.,

Hemyock, Devon. THOS. LOGAN.

SIR,—I have been disturbed, and I am sure many of my colleagues will have been, to read the letter from Mr. Philip Rhodes (October 5, p. 868), in which he suggests that a general practitioner can expect to earn £3,000 per year in his first five years in general practice. Such an event must be extremely rare. Your correspondent will surely have read the widely publicized findings of the Review Body and their proposed average payment to general practitioners of £2,750 a year. It needs only a little calculation to estimate that less than one third of all practitioners will be receiving £3,000 a year or more under the new rates which will apply, and the new entrant can hardly expect to be earning a sum of this order. I have calculated my own net earnings over the past three years, and find that the average is less than £2,200, and this after 12 years in general practice.

Your report on the findings of Dr. Annis Gillie's committee (October 5, p. 861) will, I hope, serve to further emphasize the urgent need for a further look at the present method of paying the general practitioner, and the amount of total remuneration.—I am, etc.,

S. COPP.

Cardiff.

SIR,—It is doubtful if a useful purpose can be served by quibbling over the relevant earnings of family doctors and consultants, but Mr. Philip Rhodes's letter (October 5, p. 868) was so full of incorrect conclusions that I feel the matter should be put more in perspective.

1. There are no grounds for assuming, as he appears to, that family doctors do not carry out duties as junior or even senior registrars before entering general practice.

2. On entering practice, except in the increasingly rare instances of single-handed practices, most doctors take from three to five years to achieve parity with their senior partners, and I would have thought the average earning of £3,000 a year was on the high side.

3. When the consultant enters practice his premises and equipment are provided for him. A general practitioner has to provide these at considerable capital cost, and furthermore has to maintain them to the detriment of his current income.

If these three factors are taken into consideration the alleged disparity would, I think, disappear.—I am, etc.,

R. A. KEABLE-ELLIOTT.

Stokenchurch, Bucks.

SIR,—Mr. Philip Rhodes (October 5, p. 868) has some valid points to make, but his figures are ambiguous. If the salary of a G.P. is £3,000 gross, then it is £2,100 net and the total seven and a half years' earnings are £13,500. If the salary is £3,000 net, then this indicates a gross salary of £4,285. This is, I think, rare in the first five years of partnership.—I am, etc.,

D. MORTON.

Middlesbrough, Yorks.

*** A considerable number of letters have been received expressing disagreement with Mr. Philip Rhodes's conclusions on this subject (October 5, p. 868). It is regretted that pressure on space makes it impossible for us to print more than a few of these.—ED., *B.M.J.*

Obstetrics in General Practice

SIR,—Mr. David Brown's article (September 7, p. 597) understates the enormous fillip to thought which his course at Chelmsford offers. That the hospitals may or may not obtain control of every confinement is not of paramount importance in considering the value of this course, and the type of course he envisages for postgraduate teaching centres. Rather should one appreciate what he has attained overall.

Within 24 hours of their arriving, two general practitioners (unknown hitherto to each other, nor to Mr. Brown and his staff) are smoothly absorbed into the day-to-day activities of a busy unit. At all times each practitioner is made to feel, effortlessly it seems, that he is an integral member of the unit, and practice and teaching are combined with skilful continuity. Whatever criticism may be levelled at the policy of providing general practitioners with a course in obstetrics because of trends in other countries, I would suggest that the "Chelmsford Set" have found an answer to smooth relations within the profession, well worth closer examination.

If this course does no more than reimbue us with an enjoyment of our job, and some of the confidence which most publications are attempting to destroy, then in sending us back to our practices with a little more knowledge, assurance, and, above all, enthusiasm the "Chelmsford Set" have provided a first-rate refresher course which might with advantages well become a basis for future postgraduate training.—I am, etc.,

L. H. G. OAKSHOTT.

Thorne, Yorkshire.

War Pensioners' Widows

SIR,—As chairman of the Northampton and District War Pensions Committee of the Ministry of Pensions and National Insurance and also a member of the county committee of the British Legion in Northamptonshire, I wish, through your journal, to bring to the notice of doctors a matter which is causing us increasing concern.

It is believed that a number of widows of war pensioners are losing their pension entitlement because the death certificate does not give any evidence establishing a connexion, either directly or by way of aggravation or hastening, with the pensionable disability, even when such connexion clearly exists. Specific cases of hardship could be quoted, but this letter should be as brief as possible and they are not really necessary to enable me to make my point.

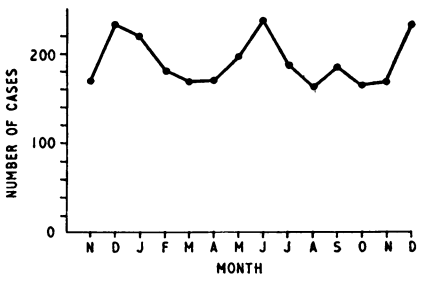
The opinion is held that the Ministry of Pensions acts more readily on the information contained in the actual certificate, and it may be very difficult to appeal if the appeal is based on "second thoughts." It has been said that it is for the relatives to remind the doctor at the time that the deceased was a war pensioner, but in the circumstances surrounding a bereavement they cannot always be relied on to do so. It is hoped, therefore, that doctors, if satisfied that a cause or connexion, however remote, exists between the pensionable disability and the condition actually causing death, will indicate this fact in order to give prima facie evidence on which the Ministry of Pensions can act or at least make further investigations.—I am, etc.,

Northampton. W. A. CLEMENTS.

Seasonal Variation in Leukaemia Incidence

SIR,—Dr. J. A. H. Lee's letter (September 7, p. 623) on the seasonal variation in leukaemia incidence is of great value to anyone interested in this subject. Apart from the points discussed by him in the text, Dr. Lee's Table I provides further information of interest. Thus, the lowermost line in this Table, the "total" adult leukaemias, shows a strikingly well-defined bi-modal seasonal distribution (see Fig.), with one peak in May–June and another equally high one in December–January. This appears to be due to the type of seasonal variation of the numerically largest (two-thirds of the total) section of adult leukaemias designated unfortunately very vaguely the "Remainder and not Stated."

In a much smaller but personally observed and therefore more fully documented series of 56 cases of acute adult



Graph of the lowermost line, "total" adult leukaemias, of Table I, from Dr. J. A. H. Lee's letter (*Brit. med. J.*, 1963, **2**, 623).

leukaemia a similar bi-modal seasonal distribution was observed.[1] The existence of two peaks was noted whether the time of clinical onset was studied, or the moment of diagnosis, or that of death. The same two peaks, one in winter and another in spring, were present when the total morbidity—i.e., the number of cases under observation at any given time—was plotted. During the discussion of our paper at the Lisbon Haematological Congress' two Continental haematologists stated that their findings were of a similar character.

The bi-modal seasonal variation of certain types of leukaemia may be fruitfully compared with that of other neoplastic diseases and diseases of viral aetiology.—I am, etc.,

A. SWAN.

Pathological Laboratory,
St. James' Hospital,
London S.W.12.

REFERENCE

1 Swan, A., Petrelli, M., and Surtees, S. J., in *Proceedings of the 9th Congress of the European Society of Haematology, Lisbon, 1963*. Karger, Basle and New York. In press.

A Dangerous Magpie

SIR,—A patient of mine, hearing her 5-month-old baby screaming in her pram in the garden, rushed out to find an adult magpie perched with a strong grip on the child's right forearm and pecking at the child's eyes. So determined was the attack that the bird was only driven off with difficulty. There were minor abrasions on the eyelids and scratches on the forearm, none of which was serious.

In 30 years' rural practice this is the first time I have ever heard of such an attack. I thought the incident might be worthy of record.—I am, etc.,

Winster, Derbyshire. A. D. STOKER.

Sodium Fluoride and Cell Growth

SIR,—As there has recently been controversy in the Oxford area regarding the addition of fluoride to the city water supply we felt it appropriate to study the effects of sodium fluoride in concentrations comparable to that proposed for the water supply upon cells in tissue culture. Two lines of mammalian cells were used: (1) HeLa S-3_{oxf}, derived from a patient with carcinoma of the cervix and acquired from Dr. Charles Ford, M.R.C. Radiobiological Research Unit, Harwell, in 1962. This line was grown in medium 199 (Glaxo) supplemented with 10% fresh human AB serum. (2) A clone of strain L mouse fibroblasts obtained from Dr. John Paul (Glasgow) and sub-cloned in this laboratory by Dr. A. H. W. Nias. These cells were grown in medium 199 supplemented with 2% pooled calf serum (Oxoid) and 0.5% Bacto-Peptone (Difco) for growth curve experiments or with 10% pooled calf serum for plating experiments. Both lines of cells were grown in sealed 12-oz. (340-ml.) bottles (medical flats), 20 ml. medium per bottle, 5×10^5 to 1×10^6 cells per bottle initial inoculum, or in sealed 6-oz. (170-ml.) bottles (medical flats), 10 ml. medium and $1–5 \times 10^5$ cells initial inoculum per bottle. Growth curves were determined by counting the number of cells per bottle after various periods of incubation at 37° C.; the medium was poured off and the cells trypsinized from the glass surface, diluted immediately 1:50 with ice-cold normal saline solution and counted in a Coulter Model D electronic cell counter which had previously been calibrated against haemacytometer counts under phase microscopy. In all experiments duplicate bottles were counted for each experimental point.

In every experiment in which sodium fluoride in concentration of 0.1 mg./l. (1/10 p.p.m.) or more was added to the medium the growth of both these cell lines was appreciably depressed (see Table). Additional sodium chloride in concentration equal to the highest dose of sodium fluoride used did not depress the growth rate.

In an attempt to determine whether the depression in growth observed in these cell lines was due to direct lethal effects, the cells were grown in clonal culture after the method of Puck *et al.*[1] In these experiments known numbers of single cells were inoculated into Falcon polystyrene 5 cm. T.C. petri dishes in appropriate medium and incubated in a humidified 5% CO–air atmosphere for 14 days. The dishes were then fixed and stained with Leishman's stain and the number of macroscopically visible clones of cells counted (clones of more than 30 cells scored as reproductively intact and viable). In these experiments *no* significant reduction of cell reproductive capacity was detected at concentrations of sodium fluoride up to 10 mg./l. (10 p.p.m.); however, at 100 mg./l. (100 p.p.m.) there was total disappearance of viable cells from the plates.

Per cent. of Control Growth in Seven Days

Cell Type	Concentration of Sodium Fluoride in Medium		
	0·1 mg./l. (1/10 p.p.m.)	1·0 mg./l. (1 p.p.m.)	10·0 mg./l. (10 p.p.m.)
Human Carcinoma (HeLa S-3_{oxf})			
Experiment 1	—	72·5	69·8
2	82·5	89·8	73·0
3	92·5	90·2	70·9
4	85·5	85·9	75·5
Average	86·8	84·6	72·3
Mouse Fibroblast (L, clone 1^2/oxf)			
Experiment 1	—	78·4	72·6
2	68·5	77·0	53·0
3	95·0	81·1	65·7
4	92·1	94·3	65·6
Average ..	85·2	82·7	64·2

The growth of two types of mammalian cells *in vitro* has thus been shown to be inhibited by extremely minute quantities of sodium fluoride in the growth medium—quantities equivalent to those recommended for use in drinking-water. As significant decrease in cell reproductive capacity does not occur until very much higher fluoride concentrations are reached, this reduction of growth is probably due to a decreased rate of cell division, not to direct and immediate cell-killing.

R. J. B. is Helen Hay Whitney Fellow in Radiobiology at Oxford University.

We should like to acknowledge the continuing support of the Damon Runyan Memorial Fund for the work of this laboratory and the enlightened guidance of Dr. Frank Ellis, Director of the Department of Radiotherapy.

—We are, etc.,

ROGER J. BERRY.
W. TRILLWOOD.

Radiobiology Laboratory,
Radiotherapy Department, and
The Pharmacy,
Churchill Hospital, Oxford.

REFERENCE

1 Puck, T. T., Marcus, P. I., and Cieciura, S. J., *J. exp. Med.*, 1956, **103**, 653.

Blood Supply of Long Bones

SIR,—In an article in the *Journal of Bone and Joint Surgery* for May, 1963, Professor J. Trueta examines the question of a possible periosteal arterial supply to the cortex of long bones.

Many of your readers will no doubt be aware that several recent investigators[1–4] have concluded that, in normal circumstances, a periosteal arterial supply to the compact bone of the shaft is negligible in amount, and that the blood flows from the medulla out through the cortex into the periosteum. In other words there is a single circulatory system in compact bone and the direction of the blood flow is centrifugal. Professor Trueta writes, however, that the outer third is supplied by periosteal arteries and the inner two-thirds by medullary arteries. This older view implies that there are two separate circulatory systems in bone cortex each possessing its own arterial supply, capillary field, and venous drainage. These peculiarities have never been demonstrated anatomically. As evidence for his statement, Professor Trueta briefly describes his experiments, of the type so beautifully executed by Professor Marneffe,[5] wherein the outer compactum survived destruction of the medullary supply, or died following suppression of the periosteal blood flow. As has been fully explained elsewhere,[6] these pathological findings are not in conflict with the known facts relating to the normal vascular anatomy of bone. In brief, these show that a unified cortical capillary field is regularly interposed between periosteal capillaries and venules externally and medullary arteries internally. Hence an anatomical basis is to hand whereby, for example, suppression of the medullary supply allows blood from the periosteum to pass into the cortex and so exercise the function of a collateral blood route to bone.